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CLINICAL REVIEW

The role of sleep hygiene in promoting public health: A review of empirical evidence

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SUMMARY

The ineffectiveness of sleep hygiene as a treatment in clinical sleep medicine has raised some interesting questions. If it is known that, individually, each specific component of sleep hygiene is related to sleep, why wouldn't addressing multiple individual components (i.e., sleep hygiene education) improve sleep? Is there still a use for sleep hygiene? Global public health concern over sleep has increased demand for sleep promotion strategies accessible to the population. However, the extent to which sleep hygiene strategies apply outside clinical settings is not well known. The present review sought to evaluate the empirical evidence for sleep hygiene recommendations regarding exercise, stress management, noise, sleep timing, and avoidance of caffeine, nicotine, alcohol, and daytime napping, with a particular emphasis on their public health utility. Thus, our review is not intended to be exhaustive regarding the clinical application of these techniques, but rather to focus on broader applications. Overall, though epidemiologic and experimental research generally supported an association between individual sleep hygiene recommendations and nocturnal sleep, the direct effects of individual recommendations on sleep remains largely untested in the general population. Suggestions for clarification of sleep hygiene recommendations and considerations for the use of sleep hygiene in nonclinical populations are discussed.

Sleep hygiene is defined as a set of behavioral and environmental recommendations intended to promote healthy sleep, and was originally developed for use in the treatment of mild to moderate insomnia [1]. During sleep hygiene education, patients learn about healthy sleep habits and are encouraged to follow a set of recommendations to improve their sleep (e.g., avoid caffeine, exercise regularly, eliminate noise from the sleeping environment, maintain a regular sleep schedule) [2]. Although research has demonstrated links between individual sleep hygiene components and subsequent sleep, evidence for the efficacy of sleep hygiene education as a treatment for insomnia has been limited and inconclusive [2–5]. Taken together, the lack of supportive data and the availability of effective, empirically supported, behavioral treatment alternatives has led to the conclusion that sleep hygiene education is ineffective as a monotherapy for insomnia [6]. Thus, we turn our attention away from sleep hygiene in the context of clinical sleep medicine, and consider its potential utility in the realm of public health where sleep hygiene is still widely used.

Sleep problems are prevalent in the global population. Throughout this manuscript, the term “sleep problems” will be used to refer generally to any combination of acute or chronic problems with prolonged sleep onset latency (SOL), excessive wake after sleep onset (WASO), short total sleep time (TST), low sleep efficiency (SE), or poor sleep quality based on subjective and/or objective assessments. We specifically do not use “sleep problems” to refer to such difficulties as symptoms of more specific clinical sleep disorders. Recent estimates suggest that over half (56%) of Americans suffered from sleep problems over the previous year, as compared to 31% of Western Europeans and 29% of Japanese [7]. Though the majority of these individuals reported functional impairment as a result of their sleep problems, most (61–79%) did not meet clinical diagnostic criteria for insomnia based on self-reported symptoms [7]. In a similar survey of adults representing 10 countries, 31.6% of participants were classified as having insomnia while an additional 17.5% of participants were classified

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Abbreviations

CBT-I	cognitive behavioral therapy for insomnia
EEG	electroencephalography
GABA	gamma-aminobutyric acid
ICU	intensive care unit
NREM	non-rapid eye movement
PSG	polysomnography
REM	rapid eye movement
SE	sleep efficiency
SOL	sleep onset latency
SWS	slow wave sleep
TST	total sleep time
WASO	wake after sleep onset

with subthreshold insomnia [8]. Sleep problems are of growing concern to global public health because poor sleep is associated with impairments in motivation, emotion, and cognitive functioning as well as increased risk for serious medical conditions (e.g., diabetes, cardiovascular disease, cancer) and all-cause mortality, even when the symptoms are below the threshold for clinical sleep disorders [9–11]. Despite the potential widespread benefit for sleep promotion, established behavioral treatments (e.g., cognitive behavioral therapy for insomnia (CBT-I)) are largely limited to individuals who qualify for, and seek, treatment from sleep medicine professionals. The present review considers the needs of the general nonclinical population, encompassing individuals with intermittent or subsyndromal sleep impairments as well as those who may meet criteria for sleep disorders, but for whom sleep treatments are unavailable or inaccessible. Such individuals may be more likely to seek assistance from primary care providers or self-help materials to manage their sleep problems, and as a result will likely be exposed to sleep hygiene recommendations which are widely used in medical settings [2,3] and are easily accessible on the internet. As described recently [12], greater emphasis on sleep health (rather than clinical sleep disorders) more closely aligns sleep research with current healthcare objectives and broadens our understanding of sleep's full spectrum of influence on population health.

Though the utility of sleep hygiene education may be limited in clinical settings, there are several reasons to consider its potential to improve sleep and promote health in the general population. In addition to being commonly used and readily available, sleep hygiene education does not require the direct involvement of a clinician and therefore can be widely disseminated to individuals not likely to seek medical treatment for their sleep problems. As a relatively inexpensive lifestyle intervention, sleep hygiene education could serve as a first-line intervention in a stepped-care model for adults who want to improve their sleep but are not likely to qualify for, or seek, more substantial clinical treatment. Sleep hygiene recommendations may be delivered via a variety of media (e.g., print- or internet-based), resulting in increased access [13]. In addition, sleep hygiene education may be a more appealing and intuitive option for the general population. For example, examination of a tailored sleep improvement plan in cancer patients revealed that when developing their individual plan, participants preferred sleep hygiene strategies over stimulus control or sleep restriction [14]. Moreover, adherence to the sleep hygiene component was relatively high and increased over time (68–78%) compared to the other treatment components [15]. However, it is important to note that individuals with undiagnosed or untreated sleep disorders may engage in poor sleep hygiene behaviors in an attempt to cope with their poor sleep (e.g., caffeine or alcohol use), and continued efforts should be made to identify these individuals and refer them for more appropriate treatments.

Recent public health campaigns have advanced general knowledge about the importance of good sleep, though they are often focused on adequate sleep duration rather than good sleep quality, and the effectiveness of these campaigns is generally unclear. Less is known regarding scientifically valid strategies by which the average person might effectively improve their sleep. Relatively few studies have investigated the efficacy of sleep hygiene interventions in nonclinical samples [16–20]. Overall, this work has provided some preliminary support for the use of sleep hygiene education in nonclinical populations, but the findings are inconsistent. Taken together with findings in clinical samples, these data raise an interesting question. If it is known that, individually, each specific component of sleep hygiene is related to sleep, why wouldn't addressing multiple individual components (i.e., sleep hygiene education) result in improved sleep? Inconsistent and uncompelling findings may be due, in large part, to the lack of a standardized approach in the application of sleep hygiene principles to clinical practice and research. As reviewed by Stepanski and Wyatt [3], definitions of sleep hygiene are inconsistent across studies, and the individual recommendations vary widely in both content and implementation. Further, these authors recommended that future research focus on establishing clear guidelines for individual behavioral and environmental aspects of sleep hygiene, rather than focusing on sleep hygiene as a comprehensive list [3]. This approach is consistent with Hauri's original recommendations to tailor sleep hygiene recommendations to fit individual needs [1], but is inconsistent with the common public health approach of providing a standard and comprehensive set of recommendations. An important next step is to consider the empirical foundation for sleep hygiene, and identify appropriate modifications to improve its delivery and efficacy in the general population.

More specifically, the current evidence base for each individual sleep hygiene recommendation should be evaluated and expanded to support further clarification of recommendations. With a particular focus on application in nonclinical populations, the present review aims to: 1) critically review the empirical evidence for individual components of sleep hygiene recommendations, identifying inconsistencies and clarifying specific guidelines for optimal sleep promotion; 2) identify gaps in the present understanding of sleep hygiene recommendations and provide suggestions for future research; and 3) identify additional conceptual and methodological issues to consider when utilizing sleep hygiene recommendations in the general population. Particular emphasis was placed on reviewing research that directly manipulated the recommended behavior by either examining the impact of the behavior or environmental factor on sleep by manipulating it (e.g., administering caffeine to a caffeine-naïve individual and observing effects on subsequent sleep) or by observing changes in sleep after the recommended behavior change was made (e.g., asking habitual caffeine users to abstain from caffeine and observing the effects on subsequent sleep). The former strategy allows for a “clean” examination of individual effects while the latter may be confounded by conceptual “noise” (e.g., tolerance, addiction, concurrent risk factors) but more closely approximates realistic circumstances in which individuals may be using sleep hygiene strategies. To maximize its relevance to the general population, when possible, the present review is focused on adults who were not specifically recruited because they suffered from clinically diagnosed sleep disorders.

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Empirical support for individual sleep hygiene recommendations

Below we provide a review of the empirical support for several of the most common sleep hygiene recommendations including caffeine consumption, smoking, alcohol use, exercise, stress, noise, sleep timing, and daytime napping. The present review is not exhaustive, but reports on representative studies, with a particular emphasis on research that examined specific parameters of recommendations (e.g., timing of behavior, amount of exposure) and research that directly evaluated the change in sleep after following sleep hygiene recommendations. As noted above, emphasis was placed on studies that 1) used nonclinical samples (i.e., did not study individuals selected for insomnia), 2) tested the isolated impact of an individual sleep hygiene component on sleep (rather than in combination with other treatments or sleep hygiene components), and 3) utilized experimental designs. Research published in languages other than English was not included. Key findings are summarized in Table 1.

Caffeine

Caffeine is the most widely used psychoactive substance in the world [21], and its stimulant properties make it a logical target for sleep promotion efforts in the general population. On a molecular level, caffeine's alerting and sleep-disruptive effects are driven by blockade of adenosine receptors in the basal forebrain and hypothalamus (see reviews of caffeine's pharmacology [22,23]). Plasma levels of caffeine peak approximately 30 min after oral administration, and the half-life of a single dose of caffeine is 3–7 h, though this is influenced by individual differences in sensitivity, metabolism, and accumulation [22,23]. For example, the half-life of caffeine has been shown to increase with age, such that the substance remains active for longer in older adults [24]. Caffeine's impact on sleep–wake physiology is well documented, but translation of its effects into clinically relevant behavioral recommendations remains less well tested.

To establish a practical, evidence-based behavioral recommendation regarding caffeine use, it is essential to consider both the

Table 1
Summary of key findings and future directions for the application of sleep hygiene to the general population.

Sleep hygiene recommendation	Summary of findings	Directions for future research
Avoid caffeine	• Caffeine administration close to bedtime disrupts sleep • Effects of caffeine on sleep show a dose–response relationship • The impact of morning and afternoon caffeine use is less clear • Harmful effects of caffeine on sleep may be limited to caffeine-sensitive individuals • Tolerance to caffeine's effects on sleep develops within days	• Morning and afternoon caffeine use • Intermittent caffeine use and the importance of day-to-day variation • Tolerance and habituation • Identification and targeting of caffeine-sensitive individuals
Avoid nicotine	• Acute and chronic nicotine administration/smoking disrupts sleep • Arousals increase temporarily during acute nicotine withdrawal • Few/limited studies suggest sleep problems associated with smoking can be resolved after cessation and withdrawal	• Longitidunal assessments of change in sleep from pre- to post-cessation • Threshold for occasional and/or light use to impact sleep • Extent to which passive smoking disrupts sleep
Avoid alcohol	• Acute alcohol administration before bed decreases SOL but increases arousal during second half of night • The effects of alcohol on sleep are dose-dependent • Tolerance to alcohol's effects on sleep occurs within days • Sleep problems increase during acute withdrawal of dependent users • Despite modest improvement, long-term sleep problems persist in abstinent former users	• Longitudinal assessments of change in sleep during use and abstinence for dependent users • Direct tests of alcohol avoidance effects on sleep in nondependent useres • The impact of afternoon and evening use on sleep • The effect of non-dependent use patterns (e.g., light/occasional, habitual weekend use) • Combined effects of alcohol with nicotine and caffeine on sleep
Exercise regularly	• Regular and/or acute bouts of exercise produce modest improvements in sleep for individuals with and without sleep complaints (though impact of exercise training on PSG-assessed sleep is less consistent) • Current evidence does not support the claim that late-night exercise disrupts sleep	• Extent to which different types, duration, and intensity level of exercise can be specified for optimal sleep improvement • Moderating effects of age, gender, and fitness level on the impact of exercise on sleep
Manage stress	• Psychosocial stress is associated with increased pre-sleep arousal and impaired sleep • Various stress management strategies have been shown to reduce pre-sleep arousal and improve sleep (most often self-reported sleep) • Individual differences influence perception of stress and coping style	• Identify patterns in individual differences to predict those whose sleep is most affected by stress • Examine the isolated benefit of stress management beyond reduction in physiological arousal • Develop process by which individuals can evaluate their own stressors and identify the most appropriate stress management technique for their needs
Reduce bedroom noise	• Nighttime noise increases arousals • Habituation to noises occurs, but EEG arousals persist • Specific noise reduction strategies have been shown to improve sleep in some environments (most often in ICU patients)	• Further test noise-attenuating strategies in home environments using objective sleep assessments • Identify individual-level factors (e.g., age) that influence preference and efficacy of specific strategies
Sleep timing regularity	• Clinical sleep treatments encourage regularity only in wake time, which is counter to some sleep hygiene recommendations to adopt regular bed- and wake-times • Irregular sleep schedules have been associated with poor sleep, but assigning regular sleep schedules to nonclinical adults has shown limited effects on sleep improvement	• Relative importance of bed- vs. wake-time regularity in nonclinical samples • Influence of moderating factors (e.g., chronotype, age) • Threshold for schedule regularity required to promote good sleep
Avoid daytime naps	• Most research suggests that daytime naps do not have a substantial impact on subjective or objective nocturnal sleep, despite sleep hygiene recommendations to avoid naps • Nap duration and timing seem to have limited effects on the relationship between napping and nocturnal sleep	• Impact of nap elimination on nocturnal sleep in habitual nappers with poor sleep • Examination of naps in the home environment rather than laboratory • Moderating effect of age

Note. EEG = electroencephalography; ICU = intensive care unit; PSG = polysomnography; SOL = sleep onset latency.

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timing and amount of caffeine consumption in daily life. Current sleep hygiene recommendations vary widely, ranging from complete abstinence to avoiding caffeine only in the afternoon or evening [3]. However, few studies have actually tested the impact of morning caffeine consumption on subsequent nighttime sleep. Landolt and colleagues administered 200 mg of caffeine early in the morning and examined its effect on polysomnographic (PSG) sleep characteristics in nine healthy, young, male participants [25]. Though salivary caffeine levels were low by bedtime, results indicated reduced TST and SE, in conjunction with a shift from lower to higher electroencephalographic (EEG) frequencies during whole-night sleep, following morning caffeine compared to placebo. Despite these intriguing results, no study has confirmed or contradicted Landolt et al.'s conclusions in the nearly 20 y since these findings were first published. One recent study examined caffeine consumed in the home environment (primarily in the morning), and found that plasma levels of caffeine and its metabolites at bedtime were not associated with PSG-assessed sleep in either people with insomnia or good sleepers [26]. Overall, though it is plausible that acute or habitual use of caffeine may impact physiological sleep–wake systems beyond the span of its half-life, replication is necessary to confirm this effect and justify a recommendation regarding morning caffeine abstinence for all individuals.

In contrast, several studies have investigated afternoon and evening caffeine use. A recent study of 12 healthy young adults administered 400 mg of caffeine in the late afternoon and evening (i.e., within the half-life of caffeine), and found that even doses ingested up to 6 h before bedtime were associated with disturbances in both subjectively and objectively assessed sleep [27]. A review of several laboratory studies of bedtime caffeine administration indicates that administration of caffeine approximately 30 min before bedtime disrupts nightly sleep by increasing SOL and decreasing TST and SE, as well as shifting sleep architecture toward lighter sleep [23]. Notably, the amount of caffeine administered in these studies was often moderate to high (up to 600 mg, equivalent to approximately 5–6 cups of brewed coffee [28]), as it was often intended to model insomnia in humans [23]. In addition, participants in these studies were often naïve to caffeine, and therefore may systematically differ from the general population of caffeine users [23]. Thus, it is reasonable to conclude that consuming large quantities of caffeine near bedtime (i.e., equivalent to several cups of coffee) is likely to disrupt sleep, but less is known about the clinical significance of low to moderate amounts of caffeine. In a direct comparison of the effects of 0, 100, 200 and 300 mg of caffeine administered shortly before bedtime, only those who received 300 mg of caffeine showed significant sleep impairments in comparison to those who received no caffeine [29]. A few other studies have examined low to moderate doses of self-administered caffeine. For example, Lloret-Linares and colleagues conducted a double-blind trial to compare the effects of one cup of caffeinated vs. decaffeinated coffee after dinner on self-reported sleep in individuals who identified themselves as caffeine-sensitive [30]. Results indicated several significant effects of caffeinated coffee on self-reported sleep quality, but these findings may not generalize to the habitually caffeinated population. In fact, recent work has identified an adenosine receptor gene associated with caffeine sensitivity, and reported that the impact of caffeine on the sleep of caffeine-insensitive individuals may be minimal [31]. Thus, individuals might consider their own caffeine sensitivity before changing caffeine intake as a means to improve sleep. Another recent randomized, double-blind study compared five days of placebo to five days of caffeine (250 mg) self-administered 0–60 min before bedtime, and measured effects on self-reported and actigraphy-assessed sleep [32]. On the first night, there was significantly greater sleep fragmentation, poorer self-reported sleep quality, and a trend toward lower SE for those in the caffeine condition relative to placebo. Beyond the first night, however, only actigraphy-assessed SE was significantly lower in the caffeine condition. The authors interpreted these changes in between-group differences over the course of just a few days as indicative of tolerance to caffeine's effects [32]. Similarly rapid tolerance to the acute sleep-disrupting effects of caffeine administration has been observed in several other laboratory studies (see review [23]), yet the role of tolerance in attenuating caffeine's influence on sleep in habitual caffeine consumers remains largely unexplored. Finally, Hindmarch and colleagues [33] utilized a cross-over design to test the impact of tea (37.5 or 75 mg of caffeine) and coffee (75 or 150 mg of caffeine) consumption on nocturnal sleep in 30 habitual caffeine users. Participants received one type of caffeinated beverage (or water) at 9:00 h, 13:00 h, 17:00 h, and 21:00 h for one day with a six day washout period between beverage conditions. Results indicated that, compared to water, caffeine consumption was associated with greater self-reported difficulty falling asleep and lower sleep quality after controlling for the previous night's sleep, though these effects were notable only for the highest caffeine condition (coffee with 150 mg caffeine). Significant effects were also observed for actigraphy-assessed TST. In comparison to no caffeine (water), low-dose caffeine conditions (tea with 37.5 or 75 mg caffeine, coffee with 75 mg caffeine) resulted in approximately 15 fewer minutes of actigraphic TST and high-dose caffeine (coffee with 150 mg caffeine) resulted in almost 45 fewer minutes of actigraphic TST. Interestingly, this reduction in TST was moderated by habitual caffeine use, such that individuals with a lower habitual caffeine intake were more sensitive to the sleep-disrupting effects of caffeine than individuals with a higher habitual caffeine intake [33]. These data further support consideration of issues of caffeine tolerance and sensitivity in research targeting samples of habitual users.

Although many studies have examined the impact of caffeine administration on subsequent sleep, very few have examined the impact of caffeine avoidance by habitual users, despite the fact that this population would be most likely to receive, and benefit from, such recommendations. Of the few available studies, the results have been inconsistent [34–36]. Hofer and Battig [35] assigned habitual caffeine users to caffeine, no caffeine, or intermittent caffeine conditions. Results suggested slightly fewer problems falling asleep on days without caffeine, but found no other effects of caffeine abstinence on self-reported sleep characteristics. Similarly, James [36] assigned habitual caffeine users to one of four groups designed to represent caffeine abstinence, acute use, withdrawal, or habitual use. After one week, the withdrawal group (six days caffeine, one day placebo) reported longer sleep duration compared to the mean of the other three groups. In a recent study, Ho and colleagues [34] assigned habitual users to one week of caffeine or abstinence and found no change from baseline self-reported or actigraphy-assessed sleep. These null or modest effects may indicate that caffeine cessation by habitual users is not very effective at improving sleep, perhaps because tolerance has developed to caffeine's sleep-disrupting effects. However, it is important to note that participants of these studies were not screened for sleep problems prior to recruitment, often because the impact of caffeine on sleep was not a primary aim of the study. The limited effects may have been observed because there was little room for improvement in sleep from baseline. This also suggests that these samples are not representative of the individuals most likely to use sleep hygiene recommendations (i.e., people with sleep problems). Additional limitations include small sample size [34], restrictive sources of caffeine (i.e., coffee only [34,35], caffeine pill [36]), and

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reliance on self-reported sleep [35,36]. Thus, at present it is difficult to interpret the implications of these findings, but future work might extend this research by investigating the short- and long-term impact of caffeine cessation in habitual users with sleep problems.

In sum, laboratory studies have demonstrated that large doses of caffeine close to bedtime have an acute disruptive effect on human sleep, but the effects of lower doses of caffeine are smaller in magnitude and less consistent. The long-term effects of caffeine abstinence in habitual caffeine consumers are not yet known, but the limited evidence to date suggests caffeine abstinence may be more beneficial for light or intermittent caffeine users than for habitual users. In order to form effective behavioral recommendations on caffeine use and sleep, future research might consider several key points. Self-administration studies in the home environment would more closely model typical use of caffeine in the general population. Careful measurement and/or manipulation of realistic timing and dosage of caffeine intake would clarify recommendations regarding caffeine dose and timing. Finally, additional research should consider the role of caffeine tolerance and its implications for sleep. For low to moderate habitual users, the daily deviation from normal caffeine intake may be more relevant to sleep than absolute amounts. Thus, behavioral recommendations may be personalized based on an individual's habits, rather than a generalized rule. Advancements in the study of caffeine and sleep will help identify the strength of their association in regular users, and inform the establishment of practical behavioral recommendations.

Nicotine

Like caffeine, nicotine promotes arousal and wakefulness, primarily through stimulation of cholinergic neurons in the basal forebrain [22]. Sleep hygiene recommendations suggest avoidance of nicotine use to promote better sleep. As reviewed by Jaehne and colleagues, nicotine, whether from cigarette smoking or administration via pill or patch, is associated with impaired sleep [37]. Specifically, nicotine use is associated with increased SOL, decreased TST, more frequent early morning awakening and suppression of rapid eye movement (REM) sleep and slow-wave sleep (SWS), though these findings are not entirely consistent across self-report and PSG assessments (see [37]). Thus, the recommendation to avoid and/or discontinue use of nicotine to improve sleep seems reasonable. However, when recommending that a nicotine-dependent individual abstain to promote better sleep, it is critical to evaluate the direct effects of smoking cessation on sleep.

In the early stages of smoking cessation, sleep complaints are very common. This is likely due to nicotine withdrawal, which results in heightened arousal and cravings. Symptoms of withdrawal peak a few days after cessation and last for 3–4 wk [38]. Increases in the frequency and duration of arousals are the most consistent sleep problems during withdrawal, as assessed by both self-report and PSG, and have been shown to increase risk of relapse in the weeks following cessation (see [37,39]). Although the acute negative impact of smoking cessation on sleep is well documented, the long-term impact of smoking cessation on sleep, and therefore, whether it is a viable sleep hygiene recommendation, is not well known. Two large cohort studies compared the sleep of non-smokers, former smokers, and current smokers and found that former smokers did not differ significantly from non-smokers in either self-reported [40] or PSG-assessed [41] sleep. These results suggest that sleep impairments associated with smoking may be resolved after cessation and long-term sleep improvements can be achieved following smoking cessation, but such conclusions are limited by the study methodologies. Both studies defined former smokers as individuals who have smoked but do not currently smoke, and additional data were not available regarding important characteristics such as the degree of past exposure and time elapsed since cessation. Moreover, sleep data prior to smoking cessation were not available for comparison in former smokers. Additional research that examines the longitudinal trajectory of sleep from pre-cessation through withdrawal and long-term assessments would help clarify the benefits of this sleep hygiene recommendation.

The current literature also revealed several additional aspects to consider in constructing a useful and valid recommendation to improve sleep with regard to nicotine use. Although general physiological tolerance to nicotine develops quickly [22] this does not appear to translate into tolerance for nicotine's sleep-disrupting effects. Whereas caffeine tolerance results in a lesser impact on sleep disturbance, data suggest that even after years of smoking, smokers experience significantly worse sleep than nonsmokers [37]. Cigarette smoke contains many chemicals other than nicotine, and it may be that other agents disrupt sleep without developing tolerance. Alternatively, the discrepancy between smokers and nonsmokers may reflect other systematic differences between these self-selected groups. Very few studies [41,42] have adjusted for relevant group differences such as age, gender, medication use, sleep apnea, and other health risk behaviors (e.g., alcohol and caffeine use, physical inactivity) known to influence sleep, so it is difficult to draw broad conclusions about the independent effect of nicotine on sleep among smokers compared to non-smokers.

Research documenting the effects of nicotine on sleep in occasional smokers or passive (secondhand) smokers is also limited. For example, Gillin and colleagues conducted a double-blind cross-over trial to examine the impact of nicotine patch administration on PSG-assessed sleep in 12 healthy nonsmokers [43]. The amount of nicotine received was equivalent to several cigarettes [22]. Compared to placebo, nicotine administration resulted in REM suppression and early awakening, with REM rebound and normalization of wake time occurring during the subsequent recovery night. Altogether, the few studies on this topic suggest that nicotine use in nondependent smokers has acute consequences similar to use in dependent smokers. Based on current data, it is not yet clear if complete abstention from nicotine is required for optimal sleep, or if there is a dose–response relationship or a threshold under which sleep would not be disturbed (e.g., after one cigarette). This information could be relevant to individuals' self-efficacy for following sleep hygiene recommendations and their willingness to adhere. Passive smoke is another way in which nonsmokers may be exposed to nicotine. Surveys suggest that secondhand smoke exposure may be linked to sleep disturbance [44,45], though one survey defined sleep disturbance broadly as reporting insufficient sleep or rest at least one day during the past month [45]. Davila and colleagues defined sleep problems more stringently using 10 questions about diagnostic criteria for sleep disorders, and found no differences in sleep between nonsmokers with and without secondhand smoke exposure [46].

In conclusion, evidence suggests that exposure to nicotine is associated with sleep problems, particularly at high doses. Recommendations to discontinue nicotine use, however, are complicated by the temporary worsening of sleep in the acute withdrawal period following cessation and the limited evidence regarding long-term benefits. Though it seems plausible that recommendations to avoid occasional and passive smoking would also be beneficial, data are limited at this time. Future systematic evaluation of the long-term impact of smoking cessation on sleep will inform behavioral recommendations, and the incorporation of strategies to help smokers overcome withdrawal-related sleep disturbance may further improve the efficacy of sleep hygiene

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recommendations regarding nicotine use. In addition, further investigation of occasional and passive smoking will clarify accurate recommendations for non-dependent smokers by evaluating factors such as timing, frequency, and type of nicotine exposure. Current sleep hygiene recommendations regarding nicotine use are not likely to be generalizable to all individuals, and therefore efforts should be made to define guidelines appropriate for individual circumstances.

Alcohol

Alcohol use is another behavior commonly discouraged in sleep hygiene education, with recommendations ranging from complete abstinence to avoidance of excessive use just before bedtime [3]. The acute effects of alcohol administration on sleep in healthy individuals are reasonably consistent and well documented. Alcohol administration near bedtime is associated with decreased SOL and increased SWS during the first part of the night. However, once the alcohol is metabolized within the first few hours of sleep, subsequent sleep becomes lighter with increases in Stage 1 and REM sleep and more arousals (see [47–49] for reviews). These effects result from alcohol's influence on several neurochemical systems (e.g., gamma-aminobutyric acid (GABA), adenosine; see [47,50]).

Despite the compelling body of work demonstrating the impact of alcohol administration on sleep, fewer studies have examined the effects of alcohol reduction on subsequent sleep that would be expected if individuals were to follow the sleep hygiene recommendation. Upon completing a one-day sleep hygiene education program designed to eliminate alcohol intake at bedtime, Morita and colleagues found a reduction in daytime sleepiness that coincided with reduction in alcohol use at bedtime [19]. However, it is unclear whether changes in sleep should be attributed to changes in alcohol use because data were not presented separately for alcohol users versus nonusers [19]. For dependent users, long-term alcohol abstinence may result in modest sleep improvement [51,52]. However, even after years of abstinence, many sleep problems may persist, including shorter sleep duration, lighter sleep, and greater sleep fragmentation (see [53]). In addition, research investigating sleep and alcohol cessation in dependent users often focuses on the acute withdrawal stage. As with caffeine, tolerance to alcohol's sleep-disrupting effects occurs within days, and sleep parameters return to baseline for healthy nondependent individuals, even at high doses of alcohol administration [47,48]. In contrast, alcohol-dependent individuals suffer from chronic sleep disturbance, which may suggest that they do not habituate to alcohol's effects on sleep or that the dosage and timing of chronic users may elicit systematically different effects than in light or nondrinkers. Chronic alcohol use may result in lasting alterations to key physiological systems which contribute to sleep regulation [54]. Further, acute nighttime withdrawal symptoms may perpetuate sleep problems resulting from acute use [55], which can exacerbate other alcohol-related withdrawal symptoms and ultimately increase risk for alcohol relapse [48].

Translation of alcohol's influence on sleep into effective sleep-promoting recommendations is somewhat complicated and requires additional attention to several key factors, such as alcohol amount and timing. To date, most evidence is based on alcohol administration within 60 min of bedtime, and few studies have examined the effect of late afternoon or early evening drinking on nocturnal sleep, although there are several circumstances in which this type of drinking may occur (e.g., consumption of alcohol during “happy hour” immediately after work or during afternoon and evening meals). Landolt and colleagues administered alcohol to 10 middle-aged men 6 h before bedtime [56]. Although breath alcohol levels had reached zero by bedtime, the reported effects on PSG-assessed sleep were similar to those found with bedtime administration. These findings were consistent with an earlier study in four healthy participants [57], but have not yet been replicated, and plausible mechanisms to explain alcohol's effect on sleep following its metabolism have not yet been clearly identified and tested.

In addition to timing, the alcohol amounts should be considered when defining behavioral guidelines. Studies in healthy adults have generally shown a dose–response relationship between the amount of alcohol consumed and sleep onset and depth, suggesting that higher doses of alcohol are associated with worse sleep (see [47–49]). Effects are typically smaller and less consistent at lower doses of alcohol, which suggests that occasional and light consumption (1–3 standard drinks [49]) may be less likely to disrupt sleep than moderate or heavy doses. Recommendations regarding absolute amounts of alcohol, however, should consider the gender of the individual as data suggest women's blood alcohol levels are significantly higher than men's after consuming the same amount and type of alcohol [58].

In sum, for non-dependent individuals, occasional consumption of alcohol (even light amounts) shortly before bedtime can impair sleep that night. The impact of afternoon or early evening alcohol use on sleep is not yet clearly understood. For alcohol-dependent individuals, chronic sleep problems are common, which are exacerbated during acute alcohol withdrawal. Thus, the effectiveness of this sleep hygiene recommendation for alcohol-dependent individuals is not well known. Additional research should consider alcohol cessation and sleep for a variety of naturalistic alcohol use patterns (e.g., dependent users, habitual weekend drinkers, daily glass of wine with dinner) to more clearly identify use patterns that increase risk for sleep problems and should be targeted in sleep hygiene recommendations. Also, future research should investigate the collective effect of alcohol use with other substances and more carefully explore their combined impact on sleep. For example, alcohol and cigarettes are often used simultaneously, and some alcoholic beverages may be combined with caffeinated beverages (e.g., coffee, soda, energy drinks) [59], making their effects on sleep difficult to predict. These aspects of alcohol use warrant further investigation in order to provide behavioral recommendations that are meaningful to a broader audience.

Exercise

Regular exercise is a common sleep hygiene recommendation, with the caveat that exercise should be avoided close to bedtime [3]. Although its mechanisms are largely unknown, exercise may improve sleep through its effects on body temperature, arousal, and/or adenosine levels [60]. Despite recommendation regarding the sleep-enhancing benefits of habitual exercise, much of the evidence is based on examination of the impact of acute bouts of exercise on a subsequent night's sleep. Two meta-analyses found that acute exercise produces modest increases in PSG-assessed TST, non-rapid eye movement (NREM) stage 2 sleep, SWS, and latency to REM sleep, as well as a small reduction in SOL [61,62], though these findings are somewhat limited due to the reliance upon young adult participants without sleep disturbances [62]. To date, only one study has examined the effects of acute exercise on sleep in adults with insomnia; in this study, an acute bout of moderate-intensity aerobic exercise performed in the late afternoon substantially improved PSG- and diary-assessed SOL and TST on the subsequent night, whereas neither high-intensity aerobic exercise nor moderate- or high-intensity resistance exercise altered sleep compared to a baseline night [63]. Taken together, this evidence suggests that an acute bout of exercise is likely to result in a modest

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improvement in the subsequent night's sleep, but this claim should be confirmed in more representative samples of individuals with nonclinical sleep complaints.

Other studies have focused on the impact of exercise training (i.e., ≥4 wk of exercise at a specific weekly dose) on sleep in various populations with sleep disturbance, most prominently in older adults. Across these studies, a moderate-sized improvement in subjective sleep quality following exercise training is the most consistent finding [64]; the few studies that assessed sleep with PSG have produced equivocal findings. Less is known regarding the effect of exercise training on sleep in healthy individuals, though a meta-analysis suggested that the effects of chronic exercise on PSG-assessed sleep in young adults without sleep disturbances were similar to those observed for acute exercise (e.g., increased TST and SWS, decreased SOL) [61]. Thus, for those with and without sleep complaints, exercise training is associated with modest improvements in sleep.

Evidence-based recommendations regarding exercise are difficult to substantiate due to its numerous components (e.g., duration, mode, intensity, timing), as well as whether an acute exercise bout or an exercise training regimen is being considered. From earlier meta-analyses, duration of exercise moderated the acute effects of exercise on TST; the largest increases in TST were seen with exercise longer than 60 min. The mode of exercise performed (e.g., walking, resistance exercise) and participant's level of fitness generally were not significant moderators [61,62]. The extent to which these findings may differ in individuals with subclinical sleep problems is not clear. Training studies have typically employed moderate-intensity aerobic exercise and/or moderate-intensity resistance exercise at doses that approximate public health guidelines; however, direct comparisons between different modes of exercise have not been performed in those with subclinical sleep problems. Direct comparisons of different doses of exercise have also been rare: Singh et al. found similar improvements in subjective sleep quality between low- and high-intensity resistance exercise in older adults [65], whereas Kline and colleagues found a dose–response relationship between the weekly duration of moderate-intensity aerobic exercise and improvement in subjective sleep quality in postmenopausal women [66].

The timing of exercise is another important factor that may impact sleep, especially in light of the common warning that exercising too close to bedtime could increase physiological arousal and disrupt subsequent sleep. However, as reviewed by Youngstedt, it is also plausible that exercising close to bedtime may improve sleep due to the acute body-heating, anxiolytic and antidepressant effects of exercise [60]. The effects of exercise on core body temperature may be especially important during the afternoon or evening, as sleep onset typically coincides with the rapid decline in body temperature [67] and exercise increases the rate of decline in body temperature by initially raising core body temperature [68]. According to a meta-analysis, exercising 4–8 h prior to bedtime has the most robust effects on subsequent sleep compared to all other times of day, including decreased PSG-assessed SOL and WASO [62]. However, studies have also found that exercising within 4 h of bedtime does not disrupt [69] or even improves subsequent sleep [70]. These results were achieved even though exercise induced increases in heart rate and core body temperature that, in some cases, remained elevated at bedtime [69]. These findings were further confirmed by a recent epidemiologic survey of 1000 adults which reported that nighttime exercise was not associated with poor sleep [71]. Thus, the available evidence does not support the claim that late-night exercise disturbs sleep in the general population.

Overall, accumulating research suggests that exercise may be a useful behavioral approach for reducing sleep disturbance. However, exercise is a complex behavior with multiple facets to consider (e.g., mode, duration, intensity, timing), and these factors have not been sufficiently examined to allow for specific behavioral recommendations regarding the impact of exercise on sleep in the general population. Future research, conducted in adults with nonclinical sleep complaints, should directly compare different modes and doses of exercise, differentiate between acute and chronic effects of exercise on sleep, and evaluate the impact of exercise timing on sleep. It will be important to explore bidirectional links between exercise and sleep, as recent data suggest that sleep may be an important predictor of physical activity participation the following day [72]. Also unknown is whether the effects of exercise on sleep are similar across genders, age groups, and differing fitness levels. These research directions will help clarify the relationship between exercise and sleep and inform evidence-based recommendations on how exercise could be optimally prescribed to improve sleep in the general population.

Stress

Although stress is not traditionally a core component of sleep hygiene, several recommendations have emerged over the years encouraging individuals to reduce worry or engage in relaxing activities, particularly right before bedtime [3]. For the purpose of the present review, the term stress refers to an event or events that lead to acute or chronic physiological (increased heart rate and blood pressure) and psychological (anxiousness, vigilance) responses. Stress can precipitate cognitive arousal (i.e., worry) and physiological arousal, which are both antithetical to problems with sleep initiation and maintenance. Indeed, numerous studies have observed an association between psychosocial stress and sleep (see [73]). Psychological stress increases psychophysiological arousal which is thought to be a primary mechanism through which stress disrupts sleep, particularly when the arousal is present at bedtime. For example, Morin and colleagues found that cognitive pre-sleep arousal (i.e., rumination before bedtime) mediated the association between daily stressors and subjective sleep quality [74]. Further, Hall and colleagues found that exposure to acute anticipatory stress close to bedtime resulted in increased sympathetic arousal, wakefulness throughout the night and less restorative sleep as measured by PSG [75]. Thus, stress management techniques (encompassing those described in sleep hygiene recommendations) have been proposed to reduce arousal associated with psychosocial stress. Designated worry time or writing a worry list has been shown to reduce sleep complaints in some individuals [76], but limited data are available to confirm these results, particularly in nonclinical samples.

Techniques known to reduce stress and arousal, such as relaxation and mindfulness-based stress reduction, have been examined in relation to sleep and have provided some preliminary support for stress management as an effective recommendation to promote sleep. A number of specific relaxation techniques are available, and most have been linked to improved sleep in individuals with insomnia (see brief review in [77–79]). Borkovec and Fowles suggested that relaxation is not directly responsible for better sleep, but rather, relaxation is focused attention that is incompatible with cognitive arousal [80]. To that end, mindfulness, which is described as focused attention on the present moment without judgment [81], has been utilized to reduce stress in multiple populations [82] and has been associated with improved subjective sleep quality. For example, in a small community sample, individuals who participated in an eight-week mindfulness meditation course which resulted in post-course improvements in self-reported sleep [83]. Similarly, college students who increased their mindfulness through a meditative movement course experienced a significant

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reduction in perceived stress, which was associated with better subjective sleep quality [84]. Mindfulness training has also successfully reduced pre-sleep arousal and worry in individuals with insomnia [85]. Overall, this work provides promising findings to support the role of stress management in improving sleep, though many of these studies relied on subjective sleep assessments. It is not yet clear whether a specific focus on stress reduction conveys any benefit to sleep above and beyond general reduction in arousal.

Perhaps more so than with any other sleep hygiene component, attention to individual differences is important for the reduction of stress-related arousal and its subsequent effects on sleep. For many individuals, the effects of acute psychosocial stress on sleep may resolve when the stressor is resolved [86,87]. However, an individual's perception of stress and coping style can exacerbate or prolong stress' impact on sleep [74,88]. For example, individuals who describe themselves as sensitive to stress had more arousals and more sleep stage transitions as measured by PSG [89]. In addition, concurrent psychopathology may further exacerbate stress and negatively influence sleep. Thus, individual circumstances will likely influence the utility of stress management recommendations. In the absence of clinical guidance, it is challenging to direct individuals toward the stress management technique most appropriate to their needs (e.g., constructive worry, relaxation, mindfulness). Future research efforts might consider the process by which the general population can evaluate their personal experience of stress and pre-sleep arousal and identify effective strategies to address them, thereby improving sleep.

Noise

Noise is a relatively clear source of sleep disturbance, and sleep hygiene recommendations frequently advise individuals to minimize noise in their sleeping environment. However, nocturnal noises within one's normal surroundings (e.g., local traffic, music, plumbing) have the potential to impact sleep, even if they are not consciously observed. The extant literature has employed a wide range of methodologies to evaluate the impact of noise during sleep. In general, nocturnal noise increases number of arousals and results in lighter sleep (increased Stage 1 and 2 and/or suppressed SWS and REM sleep; see [11,90] for reviews). Laboratory studies using PSG have reported habituation to noise exposure during sleep within a few days [91,92]. However, autonomic arousal during sleep and subjective sleep complaints have not been shown to habituate within the limited time window employed in these studies (i.e., a few weeks) [91,92]. Research suggests that the relationship between noise and sleep is moderated by characteristics of the noise itself (e.g., continuity, type, relevance) and to individual differences in noise sensitivity (see [11]). Additional research employing strict methodologies to investigate the impact of environmental noise on sleep, particularly in the natural environment, could provide useful information to help individuals identify noises that are most likely to disrupt their sleep.

Even when the source of the noise is not directly modifiable, evidence-based strategies can be used to reduce the impact of noise on sleep. Much of this work has been conducted in the context of the intensive care unit (ICU) to improve patients' sleep. As reviewed by Xie and colleagues, both sound-reducing (i.e., ear plugs) and sound-masking (i.e., white noise) strategies have been shown to improve sleep in ICU patients [93]. Additional research has investigated the efficacy of traffic noise reduction strategies in the home environment and found modest sleep improvement, including reduced SOL [94], increased SWS [94,95], and improved subjective sleep quality [95,96]. Future research on the impact of noise on sleep might continue to examine noise management strategies in environments representative of the general population, and evaluate individual differences in the preference and efficacy of various sound-attenuating strategies including important effect modifiers (e.g., age). In sum, the sleep hygiene recommendation to reduce noise in the sleeping environment appears sound, and the continued development and testing of noise management strategies will provide the tools required for individuals to comply with this recommendation.

Sleep timing regularity

Sleep hygiene recommendations often encourage regular bed- and/or wake-times which are intended to maximize the synchrony between physiological sleep drive, circadian rhythms, and the nocturnal sleep episode [3]. Homeostatic sleep drive and the circadian system work together to promote stable patterns of sleep and wakefulness [97]. Sleep duration and continuity are worsened when the primary sleep episode is advanced (i.e., shifted earlier) or delayed (shifted later) from one's habitual timing of sleep, such as changes in sleep patterns associated with jet lag or rotating shift work (see [98]). In addition, irregular bed- and wake-times increase inter-night variability in sleep timing which, in turn, results in desynchrony between sleep–wake timing and other endogenous circadian rhythms. In clinical populations, research has suggested that individuals with insomnia have more inter-night variability in sleep timing [99,100] and self-reported and actigraphy-assessed sleep characteristics [100]. Therefore, some sleep hygiene recommendations encourage regularity of sleep timing. On the other hand, current clinical sleep medicine therapies (i.e., stimulus control, CBT-I) call for regularity in wake time but permit variability in bedtime as individuals are instructed not to go to bed until they are sleepy [101]. These treatments are effective at improving sleep in clinical populations, but the isolated benefit of the sleep timing component is not known, and the extent to which a sleep timing recommendation is effective in the absence of a clinician's guidance is unclear.

Several studies of nonclinical adult populations have examined the association between sleep timing regularity and sleep. Though not entirely consistent [102], these data typically suggest that irregular sleep schedules are associated with greater daytime sleepiness [103] and worse self-reported sleep quality [104,105]. However, these data are somewhat limited by the self-report nature of both the sleep timing and sleep characteristics, which may be similarly biased by participant recall. Additional aspects of sleep timing may influence its impact on nocturnal sleep, such as differences between weekday and weekend schedules or the relative importance of regularity in wake time as compared to bedtime [106]. Future work should aim to replicate these findings using objective verification of sleep timing and sleep characteristics. Greater lifestyle regularity has also been associated with better sleep [107], though the specific contribution of sleep timing cannot be determined.

To date, only a few studies have directly tested the efficacy of this recommendation by assigning individuals to adopt a regular sleep schedule and observing the effects on subsequent sleep. Bonnet and Alter recruited 12 college students with irregular bed- and wake-times, and assigned them to a regular sleep schedule in a sleep laboratory for 38 consecutive nights, with time in bed remaining consistent with baseline [108]. Compared to the two-week baseline period, the regular sleep schedule resulted in increased self-reported awakenings with no significant changes in PSG-assessed sleep. Similarly, Takasu and colleagues assigned a rigid sleep schedule to 14 college students with irregular baseline sleep timing and found no significant changes in actigraphy-assessed sleep or self-reported sleepiness and alertness after six days [109]. Neither study recruited participants based on presence

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of sleep problems which, combined with small sample sizes, could explain these null findings. Youngstedt and colleagues recruited a sample of older adults with long self-reported sleep duration to examine the effect of a 90-min reduction of time in bed [110]. Both experimental and control groups followed a fixed sleep–wake schedule for eight weeks and results indicated that the control group (with no reduction of time in bed) experienced a significant decrease in actigraphy-assessed SE from baseline. Again, participants had no other sleep complaints at baseline. In contrast, Manber and colleagues recruited 39 college students who reported both irregular sleep schedules and excessive daytime sleepiness [111]. Participants assigned to a four-week regular sleep–wake schedule reported significantly decreased daytime sleepiness compared with controls. Authors also noted decreased SOL and increased SE from baseline to post-intervention, but these changes were not significantly different between groups.

In sum, the evidence demonstrates a clear association between sleep schedule irregularity and sleep problems, though the data in nonclinical samples are somewhat limited by their self-report nature. Research investigating the impact of changing from an irregular to a regular sleep schedule may not generalize to the population most likely to use sleep hygiene recommendations because the participants did not have any sleep complaints. Though untested, it is plausible that a dose–response relationship may exist between sleep timing regularity and sleep problems, which could help explain the discrepant benefits of sleep timing regularity between individuals with insomnia and individuals without sleep complaints. Future research should examine the validity of this sleep hygiene recommendation in samples likely to use it (i.e., individuals with nonclinical sleep complaints) using objective assessments of both sleep timing and sleep characteristics. In light of current clinical practices, bedtime regularity should not be used as a general recommendation and future research could examine the relative impact of fixing bed and/or wake times to clarify these effects in a nonclinical population.

Napping

Daytime napping has also been posited to disrupt the homeostatic sleep drive, and sleep hygiene recommendations often include the recommendation to avoid naps of greater than 30 min (see [3]). Research on the association between daytime napping and nocturnal sleep has focused primarily on older adults. Contrary to expectations, the majority of this work has identified no significant association between daytime napping and nocturnal sleep, whether assessed by self-report [112,113], actigraphy [114], or PSG [112,115]. Similarly, in a group of healthy, young and middle-aged adults, Pilcher and colleagues found no significant association between daytime napping and self-reported nocturnal sleep [116]. A few exceptions do exist, reporting that, in older adults, daytime napping is associated with more self-reported sleep problems [117] and greater actigraphy-assessed WASO and fragmentation and lower SE [113]. Typically, naps in these participants were longer than 30 min, and these data do not support a strong relationship between daytime napping and nocturnal sleep in the general population. However, these data are not entirely consistent, warranting consideration of experimental data regarding napping and nocturnal sleep.

In addition to correlational research, several studies have directly tested the impact of napping on nocturnal sleep by introducing a daytime nap (ranging from one day to one month of napping) and observing its effects on subsequent sleep. Three such studies in samples of midlife women and older adults reported no changes in PSG-assessed nocturnal sleep following daytime napping [118–120]. Similarly, Campbell and colleagues reported no significant changes in PSG-assessed sleep of older adults following a daytime nap, with the exception of a small but significant increase in SOL (from 16 to 22 min) [121]. In contrast, others have found that implementing a daytime napping schedule does negatively impact PSG-assessed sleep. Monk and colleagues compared the sleep of older adults following counterbalanced assignment to two weeks of afternoon napping for 90 min/day and two weeks of sedentary control [122]. Self-reported sleep did not differ between the two conditions, but PSG-assessed sleep following the nap condition was worse in comparison to sleep following the no-nap control. Specifically, results revealed shorter TST, lower SE, and earlier wake times following two weeks of daily afternoon napping. However, in light of more positive findings that objective evening sleepiness was decreased during the nap condition, the authors concluded that a 90-min afternoon nap would have very minimal adverse effects on nocturnal sleep of healthy older adults [122]. Only one study to date has manipulated napping in healthy young adults. Werth and colleagues compared baseline PSG-assessed sleep to nocturnal sleep following one evening nap [123]. Results indicated that napping was harmful to several characteristics of nocturnal sleep including PSG-assessed TST, SE, SWS, REM latency, spectral slow wave activity, and self-reported SOL. Thus, the data directly examining napping's effect on nocturnal sleep are inconclusive. No study to date has actually examined the effects of eliminating napping in a nonclinical sample in an attempt to improve the sleep of habitual nappers (i.e., following the sleep hygiene recommendation to avoid daytime naps), and thus direct empirical support for this recommendation is presently limited.

Specific characteristics of the nap itself may be considered in further refining sleep hygiene recommendations regarding napping. The duration of the nap may influence the extent to which it interferes with endogenous sleep rhythms. Although evidence clearly demonstrates that short naps (<30 min) are beneficial to cognitive performance, alertness, and mood (see [124,125]), this 30 min threshold may not apply to the effects of napping on nocturnal sleep. Indeed, many of the null findings reported above included naps of greater than 30 min. Pilcher and colleagues directly compared the effects of nap duration (no nap, <20 min nap, >20 min nap) for seven days and found no significant associations between any nap duration and self-reported nocturnal sleep [116]. In addition, few studies have directly evaluated nap timing. Evening naps, in particular, may be problematic if they dissipate homeostatic drive, thus interfering with nocturnal sleep. Yoon and colleagues examined evening naps in postmenopausal women and found that evening nap duration was associated with earlier wake times and more daytime napping [126]. Surprisingly, this study also found that evening nappers had higher actigraphy-assessed SE and lower WASO than non-evening nappers. Another study of older adults reported similar positive findings, such that individuals who napped during the daytime and evening had lower actigraphy-assessed SOL and WASO and higher SE than individuals who napped during the daytime only [127]. In contrast to these habitual, naturalistic naps, an assigned evening nap was shown to negatively impact PSG-assessed sleep, though this study only recorded one day of napping [123]. Overall, there are insufficient data to inform modification of recommendations regarding nap timing or duration.

Future investigations of napping and nocturnal sleep would benefit from an increased emphasis on the impact of changing napping behavior on subsequent nocturnal sleep. In addition to considering nap characteristics such as duration and timing, future work should address some methodological limitations with the present literature. The majority of this work has been performed in laboratory settings with scheduled nap opportunities. Additional research in more naturalistic settings is required and further

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investigation should consider the effects of habitual napping. Regular napping is a routine practice for many individuals (see [124,125]), but it is not yet known whether nocturnal sleep habituates to the influence of daytime napping, similar to habituation to daytime caffeine. Also, habitual nappers may have a stronger 24-h sleep drive and therefore are able to nap without impacting nighttime sleep. It is not yet known whether occasional nappers are more vulnerable than habitual nappers to the effects of napping on nocturnal sleep. As with other sleep hygiene components, individual differences are also likely to influence the consequences of daytime napping on nocturnal sleep. In particular, age is highly relevant to natural changes in circadian rhythms as well as lifestyle and health-related factors which may influence sleep timing [128]. For example, Yoon and colleagues compared napping between young and older adults and found that older adults were more likely to nap in the evening while younger adults were more likely to nap in the afternoon [129]. Further, the relationships between naps and nocturnal sleep differed by age. Older adult evening nappers had shorter SOL, earlier wake times, and earlier circadian phase compared with older adults who did not take evening naps, whereas young adults who napped in the afternoon did not differ in nocturnal sleep from young adults who did not nap in the afternoon [129]. These findings demonstrate the importance of extension of this research into a variety of populations and contexts. In sum, only limited research is available to support the common sleep hygiene recommendation to avoid naps, which may suggest that this particular recommendation is not applicable to the nonclinical population. Additional research in a broader range of contexts and populations would be required to determine if (and under which circumstances) avoidance of naps may improve sleep in the general adult population.

Conclusions

The present review evaluated the empirical support for individual sleep hygiene recommendations for adults with nonclinical sleep problems. Specifically, we performed a selective review of research investigating the impact of caffeine use, smoking, alcohol use, exercise, stress management, noise, sleep timing, and napping on nocturnal sleep characteristics. Epidemiologic and laboratory research provide some support for the relationships between individual sleep hygiene components and sleep, and each recommendation is supported by plausible physiological and psychosocial mechanisms. However, the present review identified several critical gaps in the current evidence for the use of sleep hygiene recommendations in the general population. First, direct evaluation of the effects of following sleep hygiene recommendations is scant and inconclusive for many individual recommendations. Such data are necessary to validate the extrapolation from sleep disruption studies (e.g., does administering caffeine result in impaired sleep?) to sleep hygiene recommendations (e.g., does abstaining from caffeine result in improved sleep?). Overall, it seems that simple extrapolation may not be appropriate, as effects are far more robust for experimental sleep disruption studies than for intervention studies designed to improve sleep. This may be due, in part, to the original aims of the investigators because sleep disruption studies were not typically designed to test sleep hygiene recommendations. Future work should continue to evaluate behavioral strategies to improve sleep and address methodological and sample limitations of the extant literature.

Second, current recommendations are somewhat vague and inconsistent, and the evidence is often based on extreme circumstances. For example, the most consistent finding regarding caffeine and sleep is that large doses of caffeine just before bedtime have a negative impact on sleep. However, this evidence may not apply to individuals who do not consume large amounts of caffeine near bedtime. Similarly, with regard to smoking and alcohol use, the effects of light and occasional use are far less clear than the effects of dependent use. Though both acute exercise and habitual exercise training seem to confer modest improvements in sleep, the extant literature does not provide conclusive evidence regarding how to optimize the timing, mode, and dose of exercise to enhance sleep. This does not imply that these recommendations cannot be useful for the general population, but processes should be developed to assist individuals with evaluating their specific circumstances and identifying those behaviors most likely to result in sleep disruption. Across individual recommendations, additional research to inform more specific guidelines and cover a broader spectrum of sleep hygiene behavior would be beneficial.

Finally, certain recommendations would benefit from additional guidance regarding implementation of change. For example, noise reduction and stress management techniques are encouraged, but little guidance is available regarding techniques to use in particular circumstances; consequently, it is up to individuals to identify and employ appropriate strategies. Overall, the limited support for individual sleep hygiene recommendations in the general adult population is not the result of null effects, but rather the substantial need for replication and extension of current work. Sleep hygiene education has the potential to be a key strategy for improving sleep in the general population, and future research has the potential to extend its utility and evaluate its effectiveness.

In order to maximize the utility of sleep hygiene education in the general population, future research should address several key issues. First, the field would benefit from studies of sleep hygiene recommendations that are applicable to natural behavior patterns. Most sleep hygiene recommendations draw upon research that was not intended to test the validity of behavioral recommendations to improve sleep. Thus, much of the empirical foundation for sleep hygiene recommendations in the general population has tested artificial, and often extreme, behaviors in laboratory settings. Laboratory studies have been effective in demonstrating basic effects, but this work should be expanded to examine sleep hygiene behaviors in their naturalistic context. In particular, the impact of habituation should be carefully considered, as it may inform modification of existing recommendations. For example, tolerance to the sleep-disrupting effects of acute caffeine use develops within days, so it is likely more informative to consider an individual's deviation from regular use, as opposed to their habitual daily intake. Regular use of alcohol and nicotine also results in tolerance and dependence, which may moderate their effects on sleep. Thus, different recommendations that take into account the sleep-disrupting effects of substance withdrawal might be needed for dependent and nondependent users of these substances. Future research investigating the impact of naturalistic behavior patterns on sleep will help develop sleep hygiene recommendations that are applicable and appropriate for a broader audience.

The complex interplay between behavioral and environmental patterns must also be considered in the context of sleep hygiene. Modification of one sleep hygiene behavior may lead to unintended (and sometimes undesirable) changes in other behaviors. For example, caffeine withdrawal has been associated with increased stress and decreased exercise [130], which could result in a counterproductive adverse impact on sleep. Similarly, a reduction in napping may lead to increased caffeine use to combat daytime sleepiness [124]. In contrast, some combinations of sleep hygiene components may result in more collective sleep improvement. For example, daily exercise has been shown to decrease sleep disturbance during smoking cessation [131]. Thus, it is important for future research to evaluate these interrelationships among sleep

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hygiene components as they relate to sleep disturbance. Individuals attempting to improve their sleep with sleep hygiene recommendations should be made aware of the potential benefits and consequences of modifying multiple aspects of their behavior and, ultimately, attempts should be made to develop guidelines to assist individuals in navigating the complex process of multiple behavior change. A comprehensive lifestyle approach to sleep hygiene education would also be enhanced by further investigation into other behavioral and environmental factors known to impact sleep, such as use of nighttime television and electronic devices, interpersonal environment, and use of over-the-counter sleep aids (e.g., melatonin).

In addition, as noted in the present review and others [3,11,23,37,47,124], increased attention must be dedicated to understanding individual differences in the use and effectiveness of sleep hygiene recommendations. Many factors may affect the prevalence, nature, and impact of sleep-disrupting behaviors (e.g., age, gender, genetic polymorphisms, education, comorbid health conditions, social or occupational demands). In an early, personalized approach to sleep hygiene education, Hauri proposed one-time consultations to provide 2–4 personalized sleep hygiene recommendations to adults with insomnia [76]. The majority of patients reported that they tried the personalized recommendations and, with the exception of eliminating alarm clocks, over two-thirds of patients felt the recommendations helped them sleep at one-month follow-up, and many of these perceived benefits were retained over one year. Others have implemented individualized sleep hygiene recommendations in medical patient populations and have reported improvements in subjective sleep quality [17,132] but not actigraphy-assessed sleep [17]. Identification of reliable effect modifiers in future research will be critical to identifying in whom and under what circumstances specific sleep hygiene recommendations will be most effective and will allow for the development of a personalized form of sleep hygiene education appropriate for the general population.

Finally, it is important to note that poor sleep hygiene practices may be a compensatory response to sleep problems or disorders (e.g., self-medicating with alcohol, increased use of caffeine for daytime sleepiness). If these behaviors exist solely as a direct result of the sleep problems, modification of these behaviors is not likely to resolve the underlying sleep problem. The bidirectional relationship between waking behavior and sleep is quite complex with each positioned to influence the other. Thus, it can be difficult to identify the most appropriate target for intervention. In a recent study, Irish and colleagues examined the bidirectional influence of actigraphy-assessed sleep characteristics and waking health behaviors in midlife women. Results indicated that weekly patterns of caffeine use, alcohol use, and smoking predicted some subsequent sleep characteristics, but that sleep did not predict subsequent behaviors [133]. This may suggest that targeting waking behaviors (e.g., sleep hygiene education) has more potential to impact sleep than vice versa, but conclusions are limited by the low prevalence of waking behaviors in this sample [133]. Ultimately, modification of behaviors is a reasonable approach to sleep improvement efforts, but it is important to remember that sleep will, in turn, exert influence over subsequent waking behaviors and that some sleep problems (e.g., severe insomnia, obstructive sleep apnea) will require additional forms of treatment.

In conclusion, future research should aim to substantiate and improve the efficacy of specific sleep hygiene recommendations in the general population. This is especially important given its cost-effectiveness, ease of dissemination, and accessibility. These questions are critical to public health given current trends in sleep patterns, including subclinical sleep complaints in the general population [134]. Sleep hygiene education may find a new purpose in the promotion of sleep and population health.

Practice points

1) Sleep hygiene education has the potential to address the growing public health concern of sleep complaints in the general population.
2) Although each recommendation is theoretically sound and plausible, a review of individual sleep hygiene recommendations regarding caffeine use, smoking, alcohol use, exercise, stress, noise, sleep timing, and napping revealed that empirical support for these recommendations in the general population is lacking.
3) Much of the current knowledge regarding individual components of sleep hygiene in nonclinical samples is limited to acute effects tested in laboratory environments.

Research agenda

1) Replication and extension of research demonstrating the effects of individual sleep hygiene components on objectively-assessed sleep is required, particularly in more naturalistic contexts.
2) Improved understanding of the role of habituation to sleep hygiene behaviors and their impact on sleep is an important next step toward establishing practical recommendations.
3) The complex interplay amongst sleep hygiene behaviors should be evaluated to inform development of effective, personalized strategies to maximize sleep improvement.
4) Additional consideration of behavioral and environmental factors relevant to the general population is warranted (e.g., use of television and other electronics, use of over-the-counter sleep aids, social environment).
5) Future research investigating the impact of individual differences on the use and relevance of individual sleep hygiene components will inform future efforts to develop effective personalized sleep hygiene interventions appropriate for nonclinical populations.

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References

[1] Hauri P. Sleep hygiene. In: Hauri P, editor. Current concepts: the sleep disorders. Kalamazoo, MI: The Upjohn Company; 1977. p. 21–35.
[2] Zarcone VP. Sleep hygiene. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine. 3rd ed. Philadelphia, PA: WB Saunders; 2000. p. 657–61.
*[3] Stepanski EJ, Wyatt JK. Use of sleep hygiene in the treatment of insomnia. Sleep Med Rev 2003;7:215–25.
[4] Friedman L, Zeitzer JM, Mumenthaler MS. Role of healthy sleep practices: alcohol/caffeine/exercise/scheduling. In: Sateia MJ, Buysse DJ, editors. Insomnia: diagnosis and treatment. London: Informa; 2010. p. 260–7.
[5] Riedel BW. Sleep hygiene. In: Lichstein KL, Morin CM, editors. Treatment of late-life insomnia. Thousand Oaks, CA: Sage; 2000. p. 125–46.
[6] Morgenthaler T, Kramer M, Alessi C, Friedman L, Boehlecke B, Brown T, et al. Practice parameters for the psychological and behavioral treatment of insomnia: an update. An American Academy of Sleep Medicine report. Sleep 2006;29:1415–9.
[7] Leger D, Poursain B, Neubauer D, Uchiyama M. An international survey of sleeping problems in the general population. Curr Med Res Opin 2008;24:307–17.
[8] Soldatos CR, Allaert FA, Ohta T, Dikeos DG. How do individuals sleep around the world? Results from a single-day survey in ten countries. Sleep Med 2005;6:5–13.
[9] Banks S, Dinges DF. Behavioral and physiological consequences of sleep restriction. J Clin Sleep Med 2007;3:519–28.
[10] Walker MP. The role of sleep in cognition and emotion. Ann N Y Acad Sci 2009;1156:168–97.
*[11] Zaharna M, Guilleminault C. Sleep, noise and health: review. Noise Health 2010;12:64–9.
[12] Buysse DJ. Sleep health: can we define it? Does it matter? Sleep 2014;37:9–17.
[13] Morin CM. Chronic insomnia: recent advances and innovations in treatment development and dissemination. Can Psychol 2010;51:31–9.
[14] Berger AM, Kuhn BR, Farr LA, Lynch JC, Agrawal S, Chamberlain J, et al. Behavioral therapy intervention trial to improve sleep quality and cancer-related fatigue. Psychooncology 2009;18:634–46.
[15] Berger AM, VonEssen S, Khun BR, Piper BF, Farr L, Agrawal S, et al. Feasibilty of a sleep intervention during adjuvant breast cancer chemotherapy. Oncol Nurs Forum 2002;29:1431–41.
[16] Chen PH, Kuo HY, Chueh KH. Sleep hygiene education: efficacy on sleep quality in working women. J Nurs Res 2010;18:283–9.
[17] Hudson AL, Portillo CJ, Lee KA. Sleep disturbances in women with HIV or AIDS: efficacy of a tailored sleep promotion intervention. Nurs Res 2008;57:360–6.
[18] Kakinuma M, Takahashi M, Kato N, Aratake Y, Watanabe M, Ishikawa Y, et al. Effect of brief sleep hygiene education for workers of an information technology company. Ind Health 2010;48:758–65.
[19] Morita E, Miyazaki S, Okawa M. Pilot study on the effects of a 1-day sleep education program: influence on sleep of stopping alcohol intake at bedtime. Nagoya J Med Sci 2012;74:359–65.
[20] Yngman-Uhlin P, Fernstrom A, Borjeson S, Edell-Gustafsson U. Evaluation of an individual sleep intervention programme in people undergoing peritoneal dialysis treatment. J Clin Nurs 2012;21:3402–17.
[21] Nehlig A. Are we dependent upon coffee and caffeine? A review on human and animal data. Neurosci Biobehav Rev 1999;23:563–76.
[22] Boutrel B, Koob GF. What keeps us awake: the neuropharmacology of stimulants and wakefulness-promoting medications. Sleep 2004;27:1181–94.
*[23] Roehrs T, Roth T. Caffeine: sleep and daytime sleepiness. Sleep Med Rev 2008;12:153–62.
[24] Polasek TM, Patel F, Jensen BP, Sorich MJ, Wiese MD, Doogue MP. Predicted metabolic drug clearance with increasing adult age. Br J Clin Pharmacol 2013;75:1019–28.
[25] Landolt HP, Werth E, Borbely AA, Dijk DJ. Caffeine intake (200 mg) in the morning affects human sleep and EEG power spectra at night. Brain Res 1995;675:67–74.
[26] Youngberg MR, Karpov IO, Begley A, Pollock BG, Buysse DJ. Clinical and physiological correlates of caffeine and caffeine metabolites in primary insomnia. J Clin Sleep Med 2011;7:196–203.
[27] Drake C, Roehrs T, Shambroom J, Roth T. Caffeine effects on sleep taken 0, 3, or 6 hours before going to bed. J Clin Sleep Med 2013;9:1195–200.
[28] Bunker ML, McWilliams M. Caffeine content of common beverages. J Am Diet Assoc 1979;74:28–32.
[29] Nicholson AN, Stone BM. Heterocyclic amphetamine derivatives and caffeine on sleep in man. Br J Clin Pharmacol 1980;9:195–203.
[30] Lloret-Linares C, Lafuente-Lafuente C, Chassany O, Green A, Delcey Y, Mouly S, et al. Does a single cup of coffee at dinner alter the sleep? A controlled cross-over randomised trial in real-life conditions. Nutr Diet 2012;69:250–5.
[31] Rétey JV, Adam M, Khatami R, Luhmann UFO, Jung HH, Berger W, et al. A genetic variation in the adenosine A_{2A} receptor gene (ADORA2A) contributes to individual sensitivity to caffeine effects on sleep. Clin Pharmacol Ther 2007;81:692–8.
[32] Keenan EK, Tiplady B, Priestley CM, Rogers PJ. Naturalistic effects of five days of bedtime caffeine use on sleep, next-day cognitive performance, and mood. J Caff Res 2014;4:13–20.
[33] Hindmarch I, Rigney U, Stanley N, Quinlan P, Rycroft J, Lane J. A naturalistic investigation of the effects of day-long consumption of tea, coffee and water on alertness, sleep onset and sleep quality. Psychopharmacol 2000;149:203–16.
[34] Ho SC, Chung JW. The effects of caffeine abstinence on sleep: a pilot study. Appl Nurs Res 2013;26:80–4.
[35] Hofer I, Battig K. Cardiovascular, behavioral, and subjective effects of caffeine under field conditions. Pharmacol Biochem Behav 1994;48:899–908.
[36] James JE. Acute and chronic effects of caffeine on performance, mood, headache, and sleep. Neuropsychobiology 1998;38:32–41.
*[37] Jaehne A, Loessl B, Barkai Z, Riemann D, Hornyak M. Effects of nicotine on sleep during consumption, withdrawal and replacement therapy. Sleep Med Rev 2009;13:363–77.
[38] Hughes JR. Effects of abstinence from tobacco: valid symptoms and time course. Nicotine Tob Res 2007;9:315–27.
[39] Colrain IM, Trinder J, Swan GE. The impact of smoking cessation on objective and subjective markers of sleep: review, synthesis, and recommendations. Nicotine Tob Res 2004;6:913–25.
[40] Wetter DW, Young TB. The relation between cigarette smoking and sleep disturbance. Prev Med 1994;23:328–34.
[41] Zhang L, Samet J, Caffo B, Punjabi NM. Cigarette smoking and nocturnal sleep architecture. Am J Epidemiol 2006;164:529–37.
[42] Jaehne A, Unbehaun T, Feige B, Lutz UC, Batra A, Riemann D. How smoking affects sleep: a polysomnographical analysis. Sleep Med 2012;13:1286–92.
[43] Gillin JC, Lardon M, Ruiz C, Golshan S, Salin-Pascual R. Dose-dependent effects of transdermal nicotine on early morning awakening and rapid eye movement sleep time in nonsmoking normal volunteers. J Clin Psychopharmacol 1994;14:264–7.
[44] Nakata A, Takahashi M, Haratani T, Ikeda T, Hojou M, Fujioka Y, et al. Association of active and passive smoking with sleep disturbances and short sleep duration among japanese working population. Int J Behav Med 2008;15:81–91.
[45] Sabanayagam C, Shankar A. The association between active smoking, smokeless tobacco, second-hand smoke exposure and insufficient sleep. Sleep Med 2011;12:7–11.
[46] Davila EP, Lee DJ, Fleming LE, LeBlanc WG, Arheart K, Dietz N, et al. Sleep disorders and secondhand smoke exposure in the U.S. population. Nicotine Tob Res 2010;12:294–9.
*[47] Roehrs T, Roth T. Sleep, sleepiness, and alcohol use. Alcohol Res Health 2001;25:101–9.
[48] Stein MD, Friedmann PD. Disturbed sleep and its relationship to alcohol use. Subst Abus 2005;26:1–13.
*[49] Ebrahim IO, Shapiro CM, Williams AJ, Fenwick PB. Alcohol and sleep I: effects on normal sleep. Alcohol Clin Exp Res 2013;37:539–49.
[50] Thakkar MM, Engemann SC, Sharma R, Sahota P. Role of wake-promoting basal forebrain and adenosinergic mechanisms in sleep-promoting effects of ethanol. Alcohol Clin Exp Res 2010;34:997–1005.
[51] Williams HL, Rundell Jr OH. Altered sleep physiology in chronic alcoholics: reversal with abstinence. Alcohol Clin Exp Res 1981;5:318–25.
[52] Drummond SP, Gillin JC, Smith TL, DeModena A. The sleep of abstinent pure primary alcoholic patients: natural course and relationship to relapse. Alcohol Clin Exp Res 1998;22:1796–802.
[53] Landolt HP, Gillin JC. Sleep abnormalities during abstinence in alcohol-dependent patients. Aetiology and management. CNS Drugs 2001;15:413–25.
[54] Rehm J, Room R, Graham K, Monteiro M, Gmel G, Sempos CT. The relationship of average volume of alcohol consumption and patterns of drinking to burden of disease: an overview. Addiction 2003;98:1209–28.
[55] Roehrs T, Roth T. Sleep, sleepiness, sleep disorders and alcohol use and abuse. Sleep Med Rev 2001;5:287–97.
[56] Landolt HP, Roth C, Dijk DJ, Borbely AA. Late-afternoon ethanol intake affects nocturnal sleep and the sleep EEG in middle-aged men. J Clin Psychopharmacol 1996;16:428–36.
[57] Yules RB, Lippman ME, Freedman DX. Alcohol administration prior to sleep. The effect on EEG sleep stages. Arch Gen Psychiatry 1967;16:94–7.
[58] Graham K, Wilsnack R, Dawson D, Vogeltanz N. Should alcohol consumption measures be adjusted for gender differences? Addiction 1998;93:1137–47.
[59] Istvan J, Matarazzo JD. Tobacco, alcohol, and caffeine use: a review of their interrelationships. Psychol Bull 1984;95:301–26.
[60] Youngstedt SD. Effects of exercise on sleep. Clin Sports Med 2005;24:355–65.
[61] Kubitz KA, Landers DM, Petruzzello SJ, Han M. The effects of acute and chronic exercise on sleep. A meta-analytic review. Sports Med 1996;21:277–91.

* The most important references are denoted by an asterisk.

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[62] Youngstedt SD, O'Connor PJ, Dishman RK. The effects of acute exercise on sleep: a quantitative synthesis. Sleep 1997;20:203–14.
[63] Passos GS, Poyares D, Santana MG, Garbuio SA, Tufik S, de Mello MT. Effect of acute physical exercise on patients with chronic primary insomnia. J Clin Sleep Med 2010;6:270–5.
*[64] Yang PY, Ho KH, Chen HC, Chien MY. Exercise training improves sleep quality in middle-aged and older adults with sleep problems: a systematic review. J Physiother 2012;58:157–63.
[65] Singh NA, Stavrinos TM, Scarbek Y, Galambos G, Liber C, Fiatarone Singh MA. A randomized controlled trial of high versus low intensity weight training versus general practitioner care for clinical depression in older adults. J Gerontol A Biol Sci Med Sci 2005;60:768–76.
[66] Kline CE, Sui X, Hall MH, Youngstedt SD, Blair SN, Earnest CP, et al. Dose-response effects of exercise training on the subjective sleep quality of postmenopausal women: exploratory analyses of a randomised controlled trial. BMJ Open 2012;2:e001044.
[67] Krauchi K, Cajochen C, Werth E, Wirz-Justice A. Functional link between distal vasodilation and sleep-onset latency? Am J Physiol Regul Integr Comp Physiol 2000;278:R741–8.
[68] Horne JA, Moore VJ. Sleep EEG effects of exercise with and without additional body cooling. Electroencephalogr Clin Neurophysiol 1985;60:33–8.
[69] Myllymaki T, Kyrolainen H, Savolainen K, Hokka L, Jakonen R, Juuti T, et al. Effects of vigorous late-night exercise on sleep quality and cardiac autonomic activity. J Sleep Res 2011;20:146–53.
[70] Flausino NH, Da Silva Prado JM, de Queiroz SS, Tufik S, de Mello MT. Physical exercise performed before bedtime improves the sleep pattern of healthy young good sleepers. Psychophysiology 2012;49:186–92.
[71] Buman MP, Phillips B, Youngstedt SD, Kline CE, Hirshkowitz M. Does nighttime exercise really disturb sleep? Results from the 2013 National Sleep Foundation Sleep in America Poll. Sleep Med 2014;15:755–61.
[72] Dzierzewski JM, Buman MP, Giacobbi Jr PR, Roberts BL, Aiken-Morgan AT, Marsiske M, et al. Exercise and sleep in community-dwelling older adults: evidence for a reciprocal relationship. J Sleep Res 2014;23:61–8.
*[73] Kim EJ, Dimsdale JE. The effect of psychosocial stress on sleep: a review of polysomnographic evidence. Behav Sleep Med 2007;5:256–78.
[74] Morin CM, Rodrigue S, Ivers H. Role of stress, arousal, and coping skills in primary insomnia. Psychosom Med 2003;65:259–67.
[75] Hall M, Vasko R, Buysse D, Ombao H, Chen Q, Cashmere JD, et al. Acute stress affects heart rate variability during sleep. Psychosom Med 2004;66:56–62.
[76] Hauri PJ. Consulting about insomnia: a method and some preliminary data. Sleep 1993;16:344–50.
[77] Hauri PJ, editor. Case studies in insomnia. New York, NY: Plenum; 1991.
[78] Means MK, Lichstein KL, Epperson MT, Johnson CT. Relaxation therapy for insomnia: nighttime and day time effects. Behav Res Ther 2000;38:665–78.
[79] Lichstein KL, Riedel BW, Wilson NM, Lester KW, Aguillard RN. Relaxation and sleep compression for late-life insomnia: a placebo-controlled trial. J Consult Clin Psychol 2001;69:227–39.
[80] Borkovec TD, Fowles DC. Controlled investigation of the effects of progressive and hypnotic relaxation on insomnia. J Abnorm Psychol 1973;82:153–8.
[81] Kabat-Zinn J. Mindfulness-based interventions in context: past, present, and future. Clin Psychol Sci Pract 2003;10:144–56.
[82] Grossman P, Niemann L, Schmidt S, Walach H. Mindfulness-based stress reduction and health benefits. A meta-analysis. J Psychosom Res 2004;57:35–43.
[83] Brand S, Holsboer-Trachsler E, Naranjo JR, Schmidt S. Influence of mindfulness practice on cortisol and sleep in long-term and short-term meditators. Neuropsychobiology 2012;65:109–18.
[84] Caldwell K, Emery L, Harrison M, Greeson J. Changes in mindfulness, well-being, and sleep quality in college students through taijiquan courses: a cohort control study. J Altern Complement Med 2011;17:931–8.
[85] Ong JC, Shapiro SL, Manber R. Combining mindfulness meditation with cognitive-behavior therapy for insomnia: a treatment-development study. Behav Ther 2008;39:171–82.
[86] Van Reeth O, Weibel L, Spiegel K, Leproult R, Dugovic C, Maccari S. Interactions between stress and sleep: from basic research to clinical situations. Sleep Med Rev 2000;4:201–19.
[87] Cartwright RD, Wood E. Adjustment disorders of sleep: the sleep effects of a major stressful event and its resolution. Psychiatry Res 1991;39:199–209.
[88] Sadeh A, Keinan G, Daon K. Effects of stress on sleep: the moderating role of coping style. Health Psychol 2004;23:542–5.
[89] Petersen H, Kecklund G, D'Onofrio P, Nilsson J, Akerstedt T. Stress vulnerability and the effects of moderate daily stress on sleep polysomnography and subjective sleepiness. J Sleep Res 2013;22:50–7.
[90] Muzet A. Environmental noise, sleep and health. Sleep Med Rev 2007;11:135–42.
[91] Griefahn B, Brode P, Marks A, Basner M. Autonomic arousals related to traffic noise during sleep. Sleep 2008;31:569–77.
[92] Kawada T, Xin P, Kuroiwa M, Sasazawa Y, Suzuki S, Tamura Y. Habituation of sleep to road traffic noise as determined by polysomnography and an accelerometer. J Sound Vib 2001;242:169–78.
[93] Xie H, Kang J, Mills GH. Clinical review: the impact of noise on patients' sleep and the effectiveness of noise reduction strategies in intensive care units. Crit Care 2009;13:208.
[94] Eberhardt JL, Akselsson KR. The disturbance by road traffic noise of the sleep of young male adults as recorded in the home. J Sound Vib 1987;114:417–34.
[95] Amundsen AH, Klaeboe R, Aasvang GM. Long-term effects of noise reduction measures on noise annoyance and sleep disturbance: the Norwegian facade insulation study. J Acoust Soc Am 2013;133:3921–8.
[96] Wilkinson RT, Campbell KB. Effects of traffic noise on quality of sleep: assessment by EEG, subjective report, or performance the next day. J Acoust Soc Am 1984;75:468–75.
[97] Dijk DJ, Czeisler CA. Contribution of the circadian pacemaker and the sleep homeostat to sleep propensity, sleep structure, electroencephalographic slow waves, and sleep spindle activity in humans. J Neurosci 1995;15:3526–38.
[98] Dijk DJ, Lockley SW. Integration of human sleep-wake regulation and circadian rhythmicity. J Appl Physiol 2002;92:852–62.
[99] Jefferson CD, Drake CL, Scofield HM, Myers E, McClure T, Roehrs T, et al. Sleep hygiene practices in a population-based sample of insomniacs. Sleep 2005;28:611–5.
[100] Buysse DJ, Cheng Y, Germain A, Moul DE, Franzen PL, Fletcher M, et al. Night-to-night sleep variability in older adults with and without chronic insomnia. Sleep Med 2010;11:56–64.
[101] Morin CM. Psychological and behavioral treatments for insomnia I: approaches and efficacy. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine. 5th ed. St. Louis, MO: Elsevier Saunders; 2011. p. 866–83.
[102] Gellis LA, Lichstein KL. Sleep hygiene practices of good and poor sleepers in the United States: an internet-based study. Behav Ther 2009;40:1–9.
[103] Billiard M, Alperovitch A, Perot C, Jammes A. Excessive daytime somnolence in young men: prevalence and contributing factors. Sleep 1987;10:297–305.
[104] Carney CE, Edinger JD, Meyer B, Lindman L, Istre T. Daily activities and sleep quality in college students. Chronobiol Int 2006;23:623–37.
[105] Monk TH, Buysse DJ, Billy BD, Fletcher ME, Kennedy KS, Schlarb JE, et al. Circadian type and bed-timing regularity in 654 retired seniors: correlations with subjective sleep measures. Sleep 2011;34:235–9.
[106] Soehner AM, Kennedy KS, Monk TH. Circadian preference and sleep-wake regularity: associations with self-report sleep parameters in daytime-working adults. Chronobiol Int 2011;28:802–9.
[107] Monk TH, Reynolds III CF, Buysse DJ, DeGrazia JM, Kupfer DJ. The relationship between lifestyle regularity and subjective sleep quality. Chronobiol Int 2003;20:97–107.
[108] Bonnet MH, Alter J. Effects of irregular versus regular sleep schedules on performance, mood and body temperature. Biol Psychol 1982;14:287–96.
[109] Takasu NN, Takenaka Y, Fujiwara M, Toichi M. Effects of regularizing sleep-wake schedules on daytime autonomic functions and psychological states in healthy university students with irregular sleep-wake habits. Sleep Biol Rhythms 2012;10:84–93.
[110] Youngstedt SD, Kline CE, Zielinski MR, Kripke DF, Devlin TM, Bogan RK, et al. Tolerance of chronic 90-minute time-in-bed restriction in older long sleepers. Sleep 2009;32:1467–79.
[111] Manber R, Bootzin RR, Acebo C, Carskadon MA. The effects of regularizing sleep-wake schedules on daytime sleepiness. Sleep 1996;19:432–41.
[112] Buysse DJ, Browman KE, Monk TH, Reynolds III CF, Fasiczka AL, Kupfer DJ. Napping and 24-hour sleep/wake patterns in healthy elderly and young adults. J Am Geriatr Soc 1992;40:779–86.
[113] Goldman SE, Hall M, Boudreau R, Matthews KA, Cauley JA, Ancoli-Israel S, et al. Association between nighttime sleep and napping in older adults. Sleep 2008;31:733–40.
[114] McDevitt EA, Alaynick WA, Mednick SC. The effect of nap frequency on daytime sleep architecture. Physiol Behav 2012;107:40–4.
[115] Jean-Louis G, Kripke DF, Assmus JD, Langer RD. Sleep-wake patterns among postmenopausal women: a 24-hour unattended polysomnographic study. J Gerontol A Biol Sci Med Sci 2000;55:M120–3.
[116] Pilcher JJ, Michalowski KR, Carrigan RD. The prevalence of daytime napping and its relationship to nighttime sleep. Behav Med 2001;27:71–6.
[117] Foley DJ, Vitiello MV, Bliwise DL, Ancoli-Israel S, Monjan AA, Walsh JK. Frequent napping is associated with excessive daytime sleepiness, depression, pain, and nocturia in older adults: findings from the National Sleep Foundation '2003 Sleep in America' Poll. Am J Geriatr Psychiatry 2007;15:344–50.
[118] Campbell SS, Stanchina MD, Schlang JR, Murphy PJ. Effects of a month-long napping regimen in older individuals. J Am Geriatr Soc 2011;59:224–32.
[119] Aber R, Webb WB. Effects of a limited nap on night sleep in older subjects. Psychol Aging 1986;1:300–2.
[120] Feinberg I, March JD, Floyd TC, Jimison R, Bossom-Demitrack L, Katz PH. Homeostatic changes during post-nap sleep maintain baseline levels of delta EEG. Electroencephalogr Clin Neurophysiol 1985;61:134–7.
[121] Campbell SS, Murphy PJ, Stauble TN. Effects of a nap on nighttime sleep and waking function in older subjects. J Am Geriatr Soc 2005;53:48–53.
[122] Monk TH, Buysse DJ, Carrier J, Billy BD, Rose LR. Effects of afternoon "siesta" naps on sleep, alertness, performance, and circadian rhythms in the elderly. Sleep 2001;24:680–7.

Please cite this article in press as: Irish LA, et al., The role of sleep hygiene in promoting public health: A review of empirical evidence, Sleep Medicine Reviews (2014), http://dx.doi.org/10.1016/j.smrv.2014.10.001

[123] Werth E, Dijk DJ, Achermann P, Borbely AA. Dynamics of the sleep EEG after an early evening nap: experimental data and simulations. Am J Physiol 1996;271:R501–10.
*[124] Dhand R, Sohal H. Good sleep, bad sleep! The role of daytime naps in healthy adults. Curr Opin Pulm Med 2006;12:379–82.
*[125] Takahashi M. The role of prescribed napping in sleep medicine. Sleep Med Rev 2003;7:227–35.
[126] Yoon IY, Kripke DF, Elliott JA, Langer RD. Naps and circadian rhythms in postmenopausal women. J Gerontol A Biol Sci Med Sci 2004;59:844–8.
[127] Dautovich ND, McCrae CS, Rowe M. Subjective and objective napping and sleep in older adults: are evening naps "bad" for nighttime sleep? J Am Geriatr Soc 2008;56:1681–6.
[128] Monk TH. Aging human circadian rhythms: conventional wisdom may not always be right. J Biol Rhythms 2005;20:366–74.
[129] Yoon IY, Kripke DF, Youngstedt SD, Elliott JA. Actigraphy suggests age-related differences in napping and nocturnal sleep. J Sleep Res 2003;12:87–93.
[130] Juliano LM, Griffiths RR. A critical review of caffeine withdrawal: empirical validation of symptoms and signs, incidence, severity, and associated features. Psychopharmacology 2004;176:1–29.
[131] Grove JR, Wilkinson A, Dawson B, Eastwood P, Heard P. Effects of exercise on subjective aspects of sleep during tobacco withdrawal. Aust Psychol 2006;41:69–76.
[132] Suzuki E, Tsuchiya M, Hirokawa K, Taniguchi T, Mitsuhashi T, Kawakami N. Evaluation of an internet-based self-help program for better quality of sleep among Japanese workers: a randomized controlled trial. J Occup Health 2008;50:387–99.
[133] Irish LA, Kline CE, Rothenberger SD, Krafty RT, Buysse DJ, Kravitz HM, et al. A 24-hour approach to the study of health behaviors: temporal relationships between waking health behaviors and sleep. Ann Behav Med 2014;47:189–97.
[134] Institute of Medicine. Sleep disorders and sleep deprivation: an unmet public health problem. Washington, DC: The National Academies Press; 2006.

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